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ABSTRACT

Infant Mortality and the Health of Survivors: Britain 1910-1950*

The first half of the twentieth century saw rapid improvements in the health and height of British children. Average height and health can be related to infant mortality through a positive selection effect and a negative scarring effect. Examining town-level panel data on the heights of school children I find no evidence for the selection effect but some support for the scarring effect. The results suggest that the improvement in the disease environment, as reflected by the decline in infant mortality, increased average height by about half a centimeter per decade in the first half of the twentieth century.

JEL Classification: I12, J13, N34

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Introduction

It is now well accepted that health in historical populations is reflected in physical stature and that average height is determined during infancy and childhood by some combination of nutrition and disease. A number of econometric studies have captured these effects by using per capita income as a proxy for nutrition and infant mortality as an inverse proxy for the disease environment.[1] Long run trends indicate that height in European populations increased particularly rapidly from the late nineteenth century to the middle of the twentieth century. Yet the apparent improvement in health in the first half of the twentieth century has received less attention than it deserves. In a period spanning the two world wars and the Great Depression income growth was relatively slow and yet health improved rapidly. This suggests that advances in health and height might have been boosted by a rapidly improving disease environment, as reflected in the steep downward trend in infant mortality.

The early twentieth century saw a vigorous debate about child health, nutrition and disease. Most observers saw trends and policies that reduced infant and child mortality as reflecting health gains that persisted throughout childhood and into adulthood. But to some this was not all good news. Eugenicists like Karl Pearson suggested that the fall in mortality rates for infants and young children was leading to the survival of the unfit. This debate has resonance with a recent literature that identifies the effect of infant mortality on health and height as the balance of two forces. The first is the 'selection effect' highlighted by Pearson and the eugenicists—lower infant mortality leading to less healthy and hence shorter survivors. The second is what Bozzoli et al. call the 'scarring effect', where infant mortality stands as a proxy for the general disease environment.[2] On this account lower infant mortality should be associated with better health and faster growth in childhood.

In this paper I examine the heights of school children aged 6 to 13 in English and Scottish towns during a period of rapid decline in infant mortality between 1910 and 1950. I attempt to

[1] Comprehensive surveys of this literature can be found in Steckel, 'Stature and the standard of living' and Steckel, 'Heights and human welfare'.

[2] Bozzoli et al., 'Child mortality'.

distinguish between the selection effect and the scarring effect by associating the selection effect with infant mortality in a cohort's year of birth and the scarring effect with infant mortality prevailing in the cohort's early years of childhood. I find no evidence for the selection effect but some evidence of the scarring effect. The results suggest that, on this measure alone, improvements in the disease environment accounted for an increase in the average height of children of more than half a centimeter per decade or around a quarter of the observed increase over the period.

The rest of the paper is organized as follows. The next section summarizes the historical debate and this is followed by a discussion of the possible relationships between heights and infant mortality. I then outline trends in the key variables from the late nineteenth century and some of the literature that has attempted to explain them. In the final section I present estimates from panel data on the relationship between infant mortality and heights of school children. The concluding section draws out some implications from these results for the long-term trends in health and height.

Debating Child Health

By the end of the nineteenth century it had long been recognized that the health of the working classes was seriously deficient. This was reflected in shorter stature and higher death rates at all ages among the poor, and the working class generally, than among their middle class peers. Despite considerable progress in medical science, the causal factors in poor health were still not well understood. Some observers occasionally commented that persistently high mortality, especially among infants could, in fact, be retarding the physical deterioration that might otherwise occur. Commenting in 1885 on the likely effects of sanitary reforms, the Registrar General's Superintendent of Statistics, William Ogle, observed that:

> [I]f it be true, as it undoubtedly is, that sanitation preserves a number of sickly children from death and so adds a contingent of weakly members to the adult population, it is also true that it prevents a number of children, who under the former conditions would have their strength broken and have grown up into weak adults, from undergoing this physical deterioration, and, therefore will add to the healthiness of the adult ranks. Still it is very possible that these counterbalancing influences may have been inadequate to make up

> more than partially for the first mentioned effect of sanitation, and that the mean vitality of the adult classes may really have been diminished in the way described.[3]

The degree of unfitness among working class men was brought into sharp relief when up to two fifths of those volunteering during the Boer War were rejected as unfit for active service.[4] It prompted the Balfour Government to set up the Interdepartmental Committee on Physical Deterioration to enquire into the causes of poor health.[5] In its Report, the Interdepartmental Committee took the view that health and physique was strongly influenced by conditions during infancy and childhood.[6] It summarily dismissed the idea that there was an important element of physical deterioration occurring as the result of differential fertility across social classes or in the selective mortality of infants and children. Instead, the emphasis was placed firmly on environmental factors and nutrition.[7] The most important environmental factors predisposing to disease and disability were overcrowding, sanitary conditions and air pollution. However primacy was given to the amount and quality of food available to children, especially at the younger ages.

Some of the key findings were foreshadowed in Seebohm Rowntree's survey of working class households in York in 1899. Rowntree found that 15.5 percent were living in households with incomes insufficient merely to maintain physical efficiency and a further 27.9 percent were

[3] Registrar General, 'Supplement', p. v.

[4] Over the decade 1893 to 1902, 34.6 percent of those who underwent the army medical examination were rejected and a further 3 percent were discharged on medical grounds within two years. As the volunteers were selected from the poorer sections of society this rate of unfitness is not representative of the entire male population. On the other hand, an unknown proportion of volunteers were rejected by recruiting officers without being subjected to medical inspection and so the total rejection rate could have been as high as sixty percent (Interdepartmental committee on physical deterioration, '*Report*', Appendix 1, pp. 95-97).

[5] The implications of the poor physical condition of army recruits were first drawn to public attention by Major General Sir Frederick Maurice. However, concerns about health and physical fitness were just one part of a wider public debate, sometimes known as the Campaign for National Efficiency, see Searle, '*The quest*'.

[6] In this respect it was preceded by the Royal Commission on Physical Training (Scotland), which reported in 1903.

[7] In reaching its conclusions the Committee was particularly influenced by the evidence of Dr. Alfred Eichholz who compared heights-by-age of children in different neighbourhood schools as well as providing comparisons for a variety of indicators of health and physical defects (Interdepartmental committee on physical deterioration, *Minutes of evidence*, p. 19-38 and Appendix 19).

living in what he defined as secondary poverty.[8] Rowntree identified large families as one of the main sources of poverty, and thus an even larger proportion of children than adults were living below the poverty line. Comparing families in three different types of residential location he found that by the age of 13 boys in the most prosperous district were 3.5 inches taller than those in the poorest district. At the same time infant mortality was more than 40 percent higher in the poorest than in the most well-to-do district. Rowntree commented that "[i]t is sometimes urged that although the individual suffering indicated by high infant mortality is considerable, it is not without some counterbalancing advantages, as sickly children are thus weeded out. Even if this Spartan view be accepted, it must be remembered that of those who survive, a large proportion do so only with seriously enfeebled constitutions". [9] Thus Rowntree clearly took the view that any selection effect was overwhelmed by the scarring effects of poverty.

Among the more influential contributions to the debate that followed the Interdepartmental Committee's Report (and two National Conferences) was that of Arthur Newsholme, the Medical Officer of the Local Government Board. In his 1910 Supplement to the Local Government Board Report, devoted to infant and child mortality, he specifically addressed the question of possible selection effects. Looking across counties and towns he found that infant mortality was positively correlated with mortality at ages 1-4, with a correlation coefficient across 44 counties of 0.88. He concluded that "Any selecting influence on infant mortality is thus more than counterbalanced by the operation of causes of excessive mortality after infancy," and that "these figures considered alone render doubtful the existence of any such influence under present conditions".[10] Others such as B. L. Hutchins, who made similar comparisons, came to the same view.[11]

[8] Rowntree, '*Poverty: A study*', pp. 111, 117. As a proportion of the total population of York, these figures were 9.9 percent and 17.9 percent respectively,

[9] Rowntree, '*Poverty: A study*', p. 207.

[10] Newsholme, '*Report*', p. 14.

[11] Hutchins, 'Note'.

The most well-known protagonist of the selection view in the early twentieth century was the eminent statistician Karl Pearson. Following Francis Galton he stressed the importance of heredity.[12] If health was largely inherited, then elimination of the least fit at an early age would increase the average fitness of the survivors as well as preserving racial quality in the longer run. Not surprisingly, as a statistician, he was well aware of the statistical pitfalls in identifying the selection effect. He pointed out that the sorts of correlations identified by Rowntree, and especially by Newsholme, suffer from omitted variable bias. Looking across localities, high infant mortality and higher mortality in later years of life are both the result of common environmental factors and are therefore not necessarily cause and effect. As he put it: "the only method by which data for different districts can be compared is by endeavouring to fix the nature of the environment. We want to know whether under a constant environment, the correlation between the death rates of infancy and of childhood is positive or negative".[13]

In a paper presented to the *Royal Society of London* in 1912, Pearson examined the correlation between infant mortality and mortality among those aged 1-5 years, controlling for environmental factors by using life expectancy at age 6. Using just four observations of national average data between 1831 and 1900 he claimed that the causal effect of infant mortality on child mortality was negative. He concluded thus: "there appears to me to a fair amount of evidence that Darwinism does apply to man and, and that, for a constant environment, the higher the infantile death rate, the more resistant will be the surviving child population"[14]

In fact, Newsholme had been fully aware of the need to control for the environment and in an appendix to his 1910 Report he provided correlations between changes in infant mortality and subsequent changes in mortality at ages one to five. In this analysis the negative correlation representing the selection effect could be found only for the second and (depending on the

[12] Pearson was co-founder with Galton and W. F. R Weldon of the statistical journal *Biometrika*, which he edited from its beginning in 1901 until his death in 1936. He held the Chair in Eugenics, which was associated with the Galton Laboratory, at University College, London from 1911 to 1933. Although he refused to be a member of the Eugenics Education Society, which was founded in 1907, he became founding co-editor (with E. M. Elderton) of *Annals of Eugenics* in 1925.

[13] Pearson, 'The intensity', p. 470.

[14] Ibid, p. 476.

specification) the third year of life. He suggested that this result could be rationalized "if the mortality of infancy is selective only as regards the special dangers of infancy and its influence scarcely extends beyond the second year of life, whilst the weakening effect of a sickly infancy is of greater duration".[15] In a later study, using highly differenced aggregate time series data, Elderton and Pearson obtained essentially the same result.[16]

In the following years the debate about selection effects all but died out, not least because infant mortality was falling steeply while mortality at higher ages also continued to fall.[17] Later eugenicists such as Frank White increasingly accepted that environmental influences were uppermost while still holding to the view that selection effects mattered. According to him "with infants, in particular and with very young children, at different periods and under widely different conditions, a falling death rate may be altogether deceptive as indicative of an improvement in inherent healthfulness, since the life of the most wretchedly endowed or even mortally afflicted infant can be amazingly prolonged by a sedulous system of nursing and dietary".[18] However, such views were completely overshadowed by the accumulating evidence that childhood infections and diseases had deleterious effects, not only in the succeeding years of childhood, but throughout the subsequent life-course.[19]

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[15] Newsholme, '*Report'*, p. 82.

[16] Elderton and Pearson, 'Further evidence'. These studies, particularly that of Elderton and Pearson, were subjected to a searing statistical critique by Brownlee 'The relation', who concluded that their correlations merely picked up the periodic ebb and flow of diseases affecting children in the first few years of life.

[17] Another reason is that the falling birthrate and First World War losses led to a rising tide of pronatalism, which posed problems for the eugenics movement, see Soloway, '*Demography*',, pp. 147-156.

[18] White, 'Natural and social selection', p. 100. As Searle, '*The quest'*, has pointed out, there was some revival in the eugenics movement in the depression of the 1930s when it campaigned for voluntary sterilization. But it remained very much a fringe group and it lost support in the late 1930s, partly due to hostility from the medical profession and the Catholic Church, and possibly through guilt by association with the sterilization programmes in Nazi Germany (see also Jones, '*Social hygiene*',, p. 99 and Overy, '*The morbid age*', Ch. 3).

[19] Davey Smith and Kuh, 'Does early nutrition', p. 217. One of the most important studies was that of Kermack, McKendrick and McKinlay, 'Death rates', who showed that there were strong cohort effects in mortality rates over the life cycle. Thus relative mortality rates seemed to be related more closely to the period in which the cohort grew up rather than the period during which death rates were observed. However, adult mortality rates for those born in the nineteenth century did not relate very closely the cohorts' infant mortality—suggesting that infant mortality had different effects on subsequent health. The influence of this seminal paper on the study of the childhood origins of

During the 1920s and 1930s much of the focus of political debate was on the relationship between nutrition and the health of children and this provided the underpinning for campaigns over unemployment benefit rates and family allowances.[20] In his influential book, *Food Health and Income*, Sir John Boyd Orr estimated that at least 20 percent of children were living in households with per capita incomes of less than 10 shillings per week.[21] Boyd Orr also found that children living in households below this poverty line had a food intake that was deficient in almost every constituent while those in the next income bracket suffered deficiencies mainly in certain minerals and vitamins.[22] The importance of food availability was also highlighted in a study by M'Gonigle and Kirby of families in Stockton on Tees. These authors found that those who had been removed from a slum area (which was demolished) to a new housing estate did not enjoy better health because the fall in net income and food consumption (as a result of higher rent) more than offset the effect of better housing conditions.[23]

While stressing the primacy of nutrition, these and other writers continued to acknowledge the importance of the disease environment.[24] In his second social survey of York conducted in 1936 Rowntree, while extolling the improvements in health, income, housing and public infrastructure that had occurred since 1899, still found considerable poverty and squalor. With regard to the remaining slum dwellers he commented that "the health of thousands of them is imperilled by the housing conditions under which they live".[25] Looking across 83 towns for 1929-33, the Registrar General reported that infant mortality was positively related to overcrowding and to

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adult health is discussed by Harris, 'Commentary: The child', and Davey Smith and Kuh, 'Commentary: William Oglivy Kermack'.

[20] Mayhew, 'the 1930s nutrition'; Macnicol, '*The movement*'.

[21] Boyd Orr, '*Food, health and income*', p. 27.

[22] Boyd Orr, ibid, p. 36.

[23] M'Gonigle and Kirby, '*Poverty and public health*', p. 129.

[24] Thus M'Gonigle and Kirby, ibid, p. 148, opined that "[b]ad environment must be taken seriously," but that "environment is not the only factor so operative, and possibly not the most important one."

[25] Rowntree, '*Poverty and progress*', p. 277.

latitude, even after accounting for social class.[26] Other studies found negative correlations between overcrowding and children's heights suggesting that environmental factors continued to influence both infant mortality and child health.[27]

Nevertheless, during the interwar period the focus of debate shifted towards nutrition rather than disease as the principal determinant of the health of children.[28] This is understandable in the light of the vast improvement in infant mortality that had occurred since the turn of the century. But it also reflects the progress in nutritional science, and particularly the importance of vitamins and minerals.[29] During the War and the early postwar years, with the imposition of rationing and the subsequent introduction of family allowances, even these concerns faded. Davey Smith and Kuh point out that health policy became more focused on the causes of chronic disease in later life and on curative medical services—as reflected in the founding of the National Health Service in 1948.[30] It was not until the 1980s that the importance of childhood conditions was 'rediscovered' as the debate over the childhood origins of chronic disease in later life gathered momentum.[31] In their landmark study Barker and Osmond found a strong link between local infant mortality rates in 1921-5 and mortality in 1968-78 from ischaemic heart disease, rheumatic heart disease, bronchitis and stomach cancer.[32]

Selection, Scarring and Health

Modern studies of life course epidemiology have stressed the link between health during childhood and height, which in turn depends on some combination of nutrition and disease

[26] Registrar General, '*Statistical Review'*, 1934, p. 151.

[27] See for instance, Karn, 'An analysis'.

[28] The state of the debate in the late 1930s is reflected (from different perspectives) in surveys by Cole and Cole, '*The condition of Britain*', and Vernon, '*Health in relation to occupation*'..

[29] Harris, '*The health of the schoolchild*', pp. 127-30.

[30] Davey Smith and Kuh, 'Does early nutrition', p. 222.

[31] Ibid.

[32] Barker and Osmond, 'Infant mortality'.

factors.[33] Studies for the UK have found that serious illnesses during childhood can reduce adult heights by as much as 1-2cm.[34] Studies of the links between height and diseases point to the importance repeated respiratory and gastrointestinal infections. Tanner found that British three year-olds with a history of pneumonia or bronchitis where an inch shorter than those that were free of infection; and Rona and Florey found that British children with respiratory infections in the preceding year were around 0.4 standard deviations shorter.[35] Beard and Blaser stress the effects on growth of microbial infection giving the example of bacterium *Helicobacter pylori*, which colonises the stomach and duodenum, and is associated with crowded environments and contaminated water.[36] Both respiratory and gastrointestinal infections restrict the body's ability to absorb nutrients and may also limit growth through other mechanisms.[37] Infections such as these are common both to infants and to young children, and they reflect the exposure to pathogens predisposing to a variety of infections. However, infections that are infrequent and short-lived and may provide some immunity to further attacks. It also seems likely that the incidence of disease has smaller effects in well-nourished populations than in those where limited food availability makes it more difficult to catch up after a setback caused by a bout of illness.[38]

Not surprisingly selection effects have received far less attention and have mainly been identified for cases of severe food shortage rather than for changes in the disease environment. In one interesting example Gørgens et al., found that the selection effect of the great Chinese famine of

[33] Silventoinen, 'Determinants'.

[34] Kuh and Wadsworth, 'Parental height'; Power and Manor, 'Athsma'.

[35] Tanner, 'Growth at adolescence'; Rona and Florey, 'National study of health'.

[36] Beard and Blaser, 'The ecology of height'.

[37] Restricted absorbtion of nutrients is also associated with lower levels of insulin-like growth factor (IGF I) which is strongly linked to growth, see Rogers, 'Insulin-like growth factor-I'. Chronic infection also triggers immune responses that are metabolically demanding and also elevates cortisol levels that can reduce growth by impairing protein synthesis (Crimmins and Finch, 'Infection, inflammation, height and longevity', p. 500-1).

[38] Floud etal., '*Height, health and history*', p. 250-1, provide illustrative graphs of the effects of repeated infections on growth in early childhood in developing countries. In his survey of the debate over the McKeown thesis, Harris, 'Public health', emphasizes the interaction between nutrition and the disease environment.

1959-61 increased the adult heights of survivors, who were children at the time of the famine, by 1-2cm but this was almost exactly counterbalanced by the scarring effect, which reduced their heights by a similar amount.[39]

In a recent paper, Bozzoli et al. developed a formal model of the selection and scarring effects of infant mortality on adult heights.[40] This model is illustrated in Figure 1. The Figure shows two symmetrical bell shaped curves representing the distribution of heights. The darker curve depicts a situation where infants who fail to reach a threshold level of health and of potential height do not survive. As a result the mean height of the survivors, as reflected by the vertical dark line, is to the right of the modal point of the distribution. The lighter curve represents a situation where conditions during childhood have improved. As a result, the left hand tail of the distribution is less severely truncated. This effect alone would cause the mean height of the survivors to decline as the threshold for survival has decreased. But in the case illustrated, this leftward shift of the mean relative to the mode is outweighed by a rightward shift of the whole distribution so that the new mean for the survivors is to the right of the old mean. Thus, in this example, in response to improved environmental conditions the scarring effect dominates the selection effect.

It is worth noting that when infant mortality is very high the cutoff point on the darker curve will be further to the right. Any leftward shift in this point as a result of an improvement in environmental conditions will therefore have a larger effect on the mean height of the survivors and hence the selection effect will be stronger. Assuming that a given improvement in the health environment always shifts the mode of the distribution by the same amount, this means that the selection effect is more likely to outweigh the scarring effect in conditions where the disease burden and hence infant mortality is very high. Thus there is a nonlinear, possibly U-shaped, relationship between infant mortality and height as demonstrated formally by Bozzoli et al.[41] Of course the cutoff point need not be as abrupt as it is in the illustration. But under the more

[39] Gørgens et al., 'Stunting and selection'.

[40] Bozzoli et al. 'Child mortality'.

[41] Ibid, p. 657.

plausible assumption that the selection effect of infant mortality attenuates the left hand side of the distribution over a range, the same basic idea would apply.

In their empirical work, Bozzoli et al. examine country-level data on average heights of adults born between 1950 and 1980 in Western Europe and the United States, using as explanatory variables the level of post-neonatal mortality and GDP per capita in the cohort's birth year.[42] For Western Europe and the United States there was a strong negative effect of post-neonatal mortality on adult height, while the effect of GDP per capita was insignificant. Disaggregating post-neonatal mortality by cause of death suggests that the respiratory illnesses such as pneumonia are more important than intestinal illnesses such as diarrhoea and enteritis in explaining heights. Overall the results indicate that, for relatively affluent countries with low rates of infant mortality, the scarring effect dominates the selection effect—findings that are supported by other studies.[43] However there is some evidence that for poor countries in Africa and Asia these effects appear to cancel out, suggesting that the selection effect is more powerful where infant mortality rates are more that 100 per thousand births.[44]

Infant mortality rates of more than 100 per thousand births were characteristic of most developed economies a century ago and for England and Wales they fell below this level only on the eve of the First World War (see further below). Nevertheless, research on data reaching further back in time suggests that on balance the scarring effect dominated. In their study of aggregate time series for cohorts born in the nineteenth century in France, Switzerland and England and Wales, Crimmins and Finch estimated the effects on adult heights of mortality rates at four different periods of childhood. Overall the effects were negative suggesting again that the scarring effect dominates. They concluded that "we interpret the high childhood mortality as a direct index of the high environmental exposure to infections and inflammation by the survivors in the

[42] Post-neonatal mortality is deaths between the ages of one month and one year. Bozzoli et al., ibid, use this measure in preference to total infant mortality on the grounds that variations over time in neonatal mortality (deaths within the first month) owe more to changes in pre-and peri-natal care rather than in the prevalence of infections. This is likely to be less important for the children studied below who were born before the advent of the National Health Service.

[43] See for instance, Schmidt et al., 'Height of conscripts'.

[44] Bozzoli et al., 'Child mortality', p. 661.

cohort".[45] Interestingly, for England and Wales they find that infant mortality rates had an insignificant positive effect on adult heights as well as an insignificant negative effect on mortality rates in old age. These results are suggestive of the selection effect, but in both cases they are outweighed by the larger and more significant negative effects for mortality rates later in childhood.

It is important to recognize that the two countervailing forces, selection and scarring, represent two rather different effects. In the selection effect, infant mortality simply eliminates some proportion of a birth cohort. By contrast the scarring effect represents the general disease environment facing the survivors and thus, strictly speaking, the infant mortality that is relevant to a given cohort should be that prevailing in the years of childhood subsequent to their own infancy. In the model of Bozzoli et al. the cohort's infant mortality alone captures the net effect of selection and scarring and no direct account is taken of conditions prevailing in subsequent years of childhood.[46] In the specification used by Crimmins and Finch the post-infancy mortality rates are the childhood mortality rates experienced by a given cohort at each stage of childhood. In this specification each mortality rate contains elements of both selection and scarring.[47] As a result it is not possible to separately identify these effects by associating specific coefficients with selection and others with scarring.

In the specification employed below to explain children's heights, I use the infant mortality rate at the time of the cohort's birth to represent the selection effect and the infant mortality that prevailed in the early years of that cohort's childhood to represent the scarring effect. This latter variable does not embody selection as it does not represent death rates for the cohort in question; rather it represents the disease environment faced by that cohort during childhood. Of course the infant mortality rate for a given cohort embodies an element of both selection and scarring but since infant mortality rates are so much higher than child mortality rates they embody a much

[45] Crimmins and Finch, 'Infection, inflammation, height and longevity', p. 501.

[46] Bozzoli et al., 'Child mortality'.

[47] Crimmins and Finch, 'Infection, inflammation, height and longevity'.

larger element of selection.[48] It is also worth noting that the studies referred to above use mortality rates prevailing during infancy and childhood to explain adult heights and in some cases adult death rates. Although these observations typically occur many years after childhood, no direct account is taken of conditions in the intervening years, which could well be correlated with those prevailing during childhood.[49] By contrast, the regressions presented below relate infant mortality to the heights of children, thus eliminating potentially confounding factors relating to the adult life course.

Trends in the Mortality and Height

The profiles of infant and child mortality rates for England and Wales over five year periods from 1871 are illustrated in Figure 2. In the second half of the nineteenth century there was essentially no trend in infant mortality rates, which averaged 150 per thousand live births in England and Wales and 122 per thousand in Scotland. From around the turn of the century a steep secular decline set in that saw these rates fall to below 40 per thousand by 1950 (see also Figure 3). The mortality rates for children aged 1-4, 5-9 and 10-14 are dramatically lower and fall more gradually. By the turn of the century mortality rates were less than 20 per thousand in the 1-4 age group and less than 5 per thousand in the higher age groups. An important implication is that selection effects are likely to have been far more important in the first year of life than at higher ages. This is because case fatality rates were higher for infants than for older children. Children may have suffered a significant disease burden well beyond infancy but only rarely did it result in death.

Inspection of infant mortality rates at the county level (not shown) reveals wide variations that reflect the localized nature of the factors affecting infant mortality. In 1900-2, out of 55 counties in England, Wales and Scotland, five had infant mortality rates below 100 per thousand while

[48] As Schmidt et al., 'Height of conscripts', report, the first two years of life are the most important for growth, after which the correlation between childhood height and final height becomes much stronger. However, using the 1946 UK birth cohort Wadsworth et al., 'Leg and trunk length', show that serious illness up to the age of six has an enduring effect on height.

[49] De Stavola et al. 'Statistical issues'.

another five had rates in excess of 160 per thousand.[50] Studies of the cross-sectional variation in infant mortality rates have found that these were related to many of the variables that were the subject of debate at the turn of the twentieth century. They include the well-known differences between rural and urban locations as reflected in population density and the proportion of employment in industry and mining as well as inter-urban differences in overcrowding and sanitary conditions.[51] The fact that these environmental indicators are at least as important as income and social class suggest that infant mortality was conditioned as much by the local disease environment as by factors such as nutrition.[52]

Studies of the evolution of sanitary improvement and investment in public health infrastructure point to the correlation between their rapid expansion around the turn of the century and the downturn in infant mortality.[53] Others emphasize the effects of improved milk supplies, better knowledge of hygiene and feeding methods (particularly bottle feeding) as well as the growing importance of local health services in the form of midwives and health visitors, and not least of all, the health of mothers themselves.[54] What these studies have in common is a stress on the disease environment as the chief cause of infant mortality although they focus on different types of intervention to reduce its effects.

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[50] Lee, 'Regional inequalities', p. 57. The five counties with the highest rates were Staffordshire, Warwickshire, Nottinghamshire, Lancashire and Durham, while those with the lowest rates were Hertfordshire, Norfolk, Wiltshire, Westmorland and the Highland region of Scotland.

[51] Woods et al., 'The causes'; Lee, 'Regional inequalities'; Millward and Bell, 'Economic factors', Williamson, '*Coping*'; Congdon and Southall, 'Small area variations'. In one of the earliest studies to apply regression analysis, Woolf , 'Studies of infant mortality', examined cause-specific infant mortality across County Boroughs in 1928-38. He concluded that exposure to disease, as reflected in overcrowding, and lessened resistance, as reflected in poverty, were equally important in contributing to the variation in infant mortality.

[52] Haines, 'Socioeconomic differences'; Reid, 'Locality or class?'

[53] Bell and Millward, 'Public health expenditures', p. 243. Szreter, 'The importance', has argued that public health improvements at the local level could account for a significant amount of the decline in mortality among non-infants well before the turn of the century. By contrast, Guha, 'The importance', argues that there was little change in the rate of infection but that case fatality rates fell, largely as a result of improved nutrition. It seems likely that sanitary improvements in the later nineteenth century brought some improvement in the disease environment but that the effects on infant mortality were masked by trends in urbanization and population density, which operated in the other direction.

[54] Dyhouse, 'Working class mothers'; Fildes, 'Infant feeding practices'; Millward and Bell, 'Infant mortality'.

Annual fluctuations in infant mortality for England and Wales, and also for Scotland, are illustrated in Figure 3. These series display considerable volatility from year to year—a feature that persisted throughout the first half of the twentieth century. This volatility also underlines the interpretation of infant mortality as a sensitive indicator of the disease environment rather than as reflecting slowly evolving trends in living standards. Also notable is the correlation between variations in infant mortality rates between England and Wales and Scotland which is positive (0.66) although far from perfect. In part, this reflects nationwide weather patterns, with hot and dry years tending to raise infant mortality and cold and wet years reducing them. Based on regression analysis of these effects Woods et al. suggest that the apparently anomalous rise in infant mortality in the 1890s can be explained by the sequence of hot dry summers.[55] While the prevalence of different diseases ebbed and flowed from year to year there were also distinct local variations in their intensity, as with the influenza epidemic of 1918-9.[56]

Infant mortality rates in England and Wales by broad cause of death are reported in Table 1 for the first half of the twentieth century. These and the more disaggregated statistics that underlie them show that there was a downward trend in death rates from almost every cause. These included particularly steep declines in various forms of tuberculosis as well as in infant and childhood diseases such as bronchitis and pneumonia, and diarrhea and enteritis. Sharp declines are also recorded for measles, whooping cough, influenza and a variety of stomach ailments. While medical improvements may have contributed to the decline in death from some specific causes, the cause of death evidence suggests a significant trend improvement in the general health environment facing infants and children in the first half of the twentieth century.

Unfortunately the data on children's heights is far less extensive than the data on mortality rates. Here I use the observations for individual towns and rural districts carefully collected by Bernard Harris.[57] These were obtained from the annual reports of school medical officers. The practice of measuring children dates from the establishment of the school medical service in 1908 and was

[55] Woods et al., 'The causes', p. 362.

[56] Registrar General, '*Report on the mortality*', Reid, 'The effects'.

[57] See Harris, 'The height of schoolchildren, and '*The health of the schoolchild*'.

continued into the 1950s. As average heights by age were not reported for all ages in all towns in all years, I have used regressions with locality fixed effects and with year dummies to establish the trends in heights for boys at age 6, 8, 10 and 12. The profiles of the coefficients on the year dummies shown in Figure 4 indicate remarkably strong growth in average heights among boys over four decades. Linear trends fitted to the underlying annual data give the average annual increase in height of 1.7cm per decade for six year-olds, 2.3cm per decade for eight year-olds, 2.2cm per decade for 10 year-olds and 1.7cm per decade for 12 year olds. The trends are similar for girls (not shown); the average increases per decade at the four ages are, respectively, 1.8cm, 2.3cm, 2.6cm and 2.3cm.[58]

In contrast to the fluctuations in infant mortality, the series in Figure 4 show fairly stable rates of increase in heights (except for age 10 where there are fewer underlying observations). In Figure 5, boys' heights are plotted for three different towns and ages. This reveals sharper ups and downs in the local level data, although some of this may be due to changes in the exact mean age from year to year of those who were measured. Harris examined the relationships between heights and changes unemployment for various towns, finding that cyclical effects were mainly negative but often not significant.[59]

Compared with admittedly fragmentary data for the late nineteenth century the trends displayed in Figures 4 and 5 suggest a substantial quickening in the pace of improvement in children's health in the first half of the twentieth century. Data from Christ's Hospital school indicate that over the whole period of four decades between the 1870s and 1906-8 the heights of eight year old boys increased by 2.6cm, the heights of 10 year olds also by 2.6 cm and the heights of 12 year olds by 3.5 cm.[60] On this evidence the heights of children increased by 0.5cm-1cm per decade. Statistics from army recruits (adjusted for truncation) also suggest modest growth in

[58] Boyne et al. ''Secular change', examined trends in the heights of children from 1911 to 1953, using data obtained from school medical inspections. Their measurements of increases in height by age, 'Secular change', p. 8, are highly consistent with the figures reported here.

[59] Harris, 'The height of schoolchildren', p. 37.

[60] Rosenbaum, 'A hundred years', pp. 278-9.

heights of around 0.5 cm per decade prior to the First World War.[61] Thus the heights evidence suggests a significant increase in the rate of improvement in the health of children from around the turn of the century.[62] When placed against the trends in infant mortality they are suggestive that this may have been due to improvements in the disease environment. If so, then the diminishing scarring effect must have dominated the diminishing selection effect.

Explaining the Heights of Children.

The regression analysis that follows aims to address four issues. The first is whether there is any significant effect at all of infant mortality on the subsequent height of children at different ages. The second is to see whether infant mortality at birth and in subsequent childhood years had distinct and opposite effects as the selection and scarring hypotheses would suggest. The third is whether the net effect of scarring and selection is positive or negative. And finally, if there is some net effect, how does it contribute to the trend in the heights of children during the first half of the twentieth century?

To answer these questions I use as the dependent variable the data on average heights by age, sex, locality and year collected by Bernard Harris.[63] I have excluded from this panel dataset towns or localities for which the run of years is very short or for which infant mortality data was not available as well as counties or rural districts that embrace a heterogeneous mix of localities. Table 2 lists the 20 towns that are included in the estimation, with the number of usable observations. For each town there is a roughly equal balance of boys and girls at a range of different ages. While each average height observation is for a specific year of age the exact

[61] Ibid, p. 282.

[62] This is also the impression gained from other figures presented by Rosenbaum and earlier by Karn, 'Summary of results', and Clements, 'Changes in the mean'. For an even longer-term perspective see Floud et al., '*Height*', p. 166.

[63] Using town-level averages for height has advantages and disadvantages. One advantage is that the genetic component of height, which is the largest single influence on the variation in height across individuals, is largely averaged out. The disadvantage is that observed heights can be related only to the locality and not to household- or family-level variables. For a study of the heights of individual children during the interwar period, see Hatton and Martin, 'fertility decline'.

average age is typically somewhat higher. The exact average age is either that recorded in the original medical officer's report or is estimated by Harris. In the regressions, the fraction of a year represented by difference between the benchmark age and the estimated average age is included as a separate variable: 'part year'.

Figures for infant mortality come from the same database. However, since we need infant mortality in the years preceding the year in which height is observed the database has been augmented with additional figures taken from the Annual Reports of the Registrar General for England and Wales and the Registrar General for Scotland. For major Scottish towns infant mortality rates can be found back to the turn of the century but for England and Wales they are reported only at the country level prior to 1911. Because infant mortality is included as a lagged variable, the data is very thin for the earlier years and so I use observations for the years 1920 to 1950. The infant mortality rates are used to construct two variables. One is for infant mortality in the year of birth, which represents the selection effect. The other is average infant mortality for a period after the birth year but before the year of observation, which represents the scarring effect. For 6 to 9 year olds I used the average infant mortality rate when the cohort was aged 2-4 and for 10 to 13 year olds the infant mortality rate when the cohort was aged 2-6.

The results for the children aged 6 to 9 are reported in Table 3, where the dependent variable is height in centimeters.[64] These are fixed effects estimates, with a separate effect for each town by sex (and hence there is no single coefficient for sex). Column (1) includes only the infant mortality rate in the year of birth. This produces a coefficient that is negative, but small and insignificant. When the average infant mortality rate at ages 2-4 is added to the regression (Column 2), this produces a negative and significant coefficient, which provides support for the scarring effect. In the presence of the scarring effect, the selection effect becomes even less significant and it is dropped from the regression in column (3). The coefficient on the average infant mortality when aged 2 to 4 (column 3) implies that a fall in infant mortality of 10 per

[64] An alternative would be to take the log of height as the dependent variable, so that the coefficients could then be interpreted as proportional effects. This produced results that are qualitatively similar to those reported in Tables 3 and 4.

thousand births would increase the heights of 6-9 year-olds by a little over quarter of a centimeter.

The other coefficients are also of interest. In column (3) the coefficient on the time trend implies a secular increase in the heights of these children of about 1.8 cm per decade. The dummies for each year of age (with age 6 as the excluded group) imply growth of between four and six centimetres per year of age. Lastly the age adjustment, which is the proportion of a year by which average age exceeds the benchmark age, takes a coefficient of 4.6, which is broadly consistent with the gaps between each benchmark year of age.

Table 4 reports a similar set of regressions for the 10-13 year-olds. When entered alone, the infant mortality rate in the year of birth takes a negative but insignificant coefficient. In column (2) the average infant mortality rate over the ages 2-6 is negative and significant. As with the younger children, there is no support for the selection effect but some evidence in favour of the scarring effect. Although the latter is marginally less significant for those aged 10-13, the size of this effect is almost identical to that reported for the younger children in Table 3. For the 10-13 year-olds, the result in column 3 also indicates a secular increase in heights of about 2.3cm per decade, while the year of age dummies indicate growth of four to six centimeters per year of age.

Two further points are worth noting. First, I experimented with two alternative versions of the variable representing the scarring effect. Using the average infant mortality rate when the cohort was aged 3 to 5 (for 6-9 year-olds) and 3-7 (for 10-13 year olds) yielded similar but somewhat weaker scarring effects. Using the average infant mortality rate over the three years or the five years preceding the year of observation produced insignificant coefficients. This suggests that the scarring effects were most important during early childhood. As mentioned earlier, those effects seem to have persisted throughout the later years of childhood and into adolescence.

Second, the use of fixed effects means that the coefficients on infant mortality are estimated on the variation over time within each town and not on the variation between towns. Dropping the fixed effects from the regression in column (1) of Table 3 produces a coefficient on infant mortality of -0.04 (‘t’= 11.3); for the specification in column (3) it produces a coefficient of -0.06 (‘t’= 15.2). Thus Pearson was right to argue that exploiting the cross-sectional variation in

infant mortality leads to negative bias in the relationship between infant mortality and subsequent health. However, the bias is not large enough to produce the positive selection effect that he would have predicted under fixed effects.

A further issue is whether the effects of infant mortality varied across different groups. As the effects of infant mortality are similar for younger and older children the regressions in Table 5 combine all the years of age that are included in Tables 3 and 4. Here, infant mortality is that prevailing when the cohort was aged 2-4 and infant mortality during the birth year is dropped. Comparing columns (1) and (2), the negative effect of infant mortality is stronger for girls than for boys, which may suggest greater protection for boys, although the difference between these coefficients is not significant ('t' = 1.22). But there is also evidence of somewhat flatter height by age profile for boys and a slightly greater increase with time.

Another possibility is that the effects different across towns, perhaps as a result of differences in economic conditions or in the provision of health services.[65] Columns (3) and (4) of Table 5 compare northern towns, including those in Scotland and Wales, with those in the south. The effect of infant mortality is smaller in the north, but again the difference is not significant ('t' = 0.86). And while the trend is somewhat stronger for the north, the height by age profile is somewhat flatter. Other comparisons, not reported in Table 5 yield similar results. Splitting the towns by population size produces a coefficient on infant mortality of -0.038 ('t' = 3.62) for towns larger than a hundred thousand in 1931 and a coefficient of -0.025 ('t' = 2.91) for smaller towns. Alternatively, splitting the towns evenly between high wage and low wage (as reflected by builders labourers' wage rates in 1929) produced a coefficient of -0.023 ('t' – 2.98) for high wage and -0.032 ('t' = 2.44) for low wage towns. Overall these results suggest that, while there is some heterogeneity in the effects of the disease environment on height, the effect is always negative and the differences are not significant enough to support strong inferences.

One final question is what these results imply for the net contribution of the changing disease environment, as represented by infant mortality, to the trends in heights that are observed in

[65] Levene et al., 'Patterns of municipal health expenditure', identify differing trends in health expenditures by region and between larger and smaller towns.

these data. Over the 40 years from 1901-5 to 1941-5 infant mortality in England and Wales fell by 88 per thousand or by 22 per thousand per decade (Figure 2). Applying the coefficient in column (3) of Table 3 suggests that this would have increased the heights of 6-9 year olds (with a lag of 3-6 years) by 88×0.025 = 2.24cm, or 0.55 cm per decade. Since the average height in this age group increased over the period by about 2.1cm per decade, the scarring effect would account for about a quarter of the increase in height. For those aged 10-13 where heights increased by a little more, the scarring effect accounts for a slightly smaller share of the total growth.

It is interesting to compare these effects with those reported for by Bozzoli et al. from country-level panel data. For birth cohorts from 1950 onwards in developed countries they estimate that a fall in post-neonatal mortality of one per thousand increased adult height by 0.06cm.[66] This coefficient is twice as large as those reported here in Tables 3 and 4. However, the absolute fall in post-neonatal mortality is smaller than that of infant mortality, of which it forms part. In England and Wales post-neonatal mortality fell by 44 per thousand births from 1901-5 to 1941-5. Using the coefficient estimated by Bozzoli et al., this would imply an increase in heights of 44 × 0.06 = 2.6cm, or 0.7cm per decade. Although this coefficient applies to adult heights, it is consistent with the predictions from using infant mortality on the data for children in British towns in the years before mid-century.

Conclusion

In the context of the debate after the turn of the twentieth century it appears that the medical experts and social investigators were right to emphasise the scarring effects of the disease environment on children's health, and to discount the selection effect. There is no support for Pearson's position that some positive selection effect on health can be identified once the health environment in post-infancy childhood is taken into account. Nevertheless Pearson was right to argue that cross-sectional correlations would tend to overstate the magnitude of the scarring effect. One might also speculate that, if similar data were available for the period of higher infant

[66] Bozzoli, et al., 'Child mortality', p. 651.

mortality in the late nineteenth century, then the selection effect might have been a little stronger. But in my view it is doubtful that selection would ever have dominated scarring.

Perhaps of greater historical significance is explaining the apparently rapidly improving health of children in the first half of the twentieth century. In a recent paper Richard Martin and I analysed individual-level data from the records of Boyd Orr's investigation of the health of children in 1937-9. We found that age-standardized heights were strongly positively related to household per capita income and, in addition, we found a negative effect of family size on height. Mapping this into trends over time we found that these effects could account for a total increase in the height of 8 year-olds of about 2.6cm between 1906 and 1938, or about 40 percent of the total increase. The results presented here in Tables 3 and 4 imply that the fall in infant mortality increased heights in England and Wales by 2.0cm over the same period. These effects are clearly not mutually exclusive, as some of the decline in infant mortality would ultimately be due to increasing family incomes and falling family size.[67] Nevertheless the results do underline the fact that improvements both in nutrition and in the disease environment contributed to the trend increase in the heights of children.

Finally, it is worth commenting briefly on what the effects presented here imply for the future life course of those who were growing up during the first half of the twentieth century. It is fairly well established that height is positively associated with longevity, something that is observed in a number of British cohort studies. However this effect is not causal; both height and longevity are independently influenced by conditions during childhood. One recent study of the Boyd Orr Cohort found that infant mortality in the locality during an individual's childhood had a discernible positive influence on subsequent mortality risk. Direct experience of infection during childhood had an even stronger correlation with mortality risk and this largely obscures the background effect of the disease environment.[68] Similarly, evidence from the 1958 birth cohort indicates that children with health conditions during childhood had poorer health and lower

[67] Rising food consumption per capita is estimated to account for an increase in height of 1.6cm over this period, which is considerably less than the income effect. If view this as the pure nutrition effect, and the contribution of infant mortality as the disease effect, then together these would add 3.6cm to height between 1906 and 1938, or about three fifths of the total increase.

[68] Frijters et al. Childhood economic conditions'.

educational attainment and social status as adults.[69] Thus the evidence suggests that the disease environment experienced during childhood that is reflected in height also influenced health during the subsequent life course. As a result, conditions during first half of the twentieth century have had persistent effects on a range of health-related outcomes right up to the present.

[69] Case et al., 'The lasting impact'.

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Table 1: Cause-Specific Infant Mortality in England and Wales, 1891-1949

	1891-1900	1901-1910	1911-1920	1921-1930	1931-1940	1940-1949
All causes	153.33	127.55	100.43	71.86	58.57	44.64
Common infectious diseases	10.03	8.07	6.39	4.29	3.63	2.04
Tuberculous diseases	7.85	5.17	2.58	1.22	0.70	0.43
Diarrhoea and enteritis	25.17	20.47	14.70	7.22	5.28	4.40
Developmental and wasting diseases	44.95	43.34	37.13	30.14	26.86	20.17
Miscellaneous diseases	65.33	50.48	39.76	28.98	22.10	17.55

Source: Registrar General for England and Wales, *Statistical Review* (Part 1), 1931, 1941 and 1949.

Table 2: Towns Included in the Analysis of Heights, with Number of Observations

Town	Age 6-9	Age 10-13	Town	Age 6-9	Age 10-13
Aberdeen	40	40	Huddersfield	64	56
Abertillery	70	70	Leeds	130	114
Bath	32	32	Liverpool	44	40
Blackburn	99	82	Reading	36	30
Bradford	118	122	Rhondda	234	190
Cambridge	72	68	Sheffield	132	126
Croydon	152	152	Spenborough	132	101
Edinburgh	38	42	Stirling	20	16
Gateshead	18	18	Wakefield	90	106
Glasgow	110	82	Warrington	186	100

Table 3: Explaining the Heights of 6-9 Year Olds

	(1)	(2)	(3)
Infant mortality in birth year	-0.004	-0.000	
	(0.70)	(0.05)	
Infant mortality when aged 2-4		-0.026	-0.026
		(2.93)	(2.90)
Year	0.216	0.177	0.177
	(11.94)	(7.02)	(7.43)
Age 7	5.719	5.769	5.768
	(22.32)	(22.77)	(22.46)
Age 8	11.485	11.567	11.566
	(35.49)	(35.34)	(35.53)
Age 9	15.590	15.742	15.741
	(71.33)	(71.89)	(74.04)
Part year	4.614	4.602	4.602
	(9.89)	(9.63)	(9.62)
R^2 (within)	0.856	0.857	0.857
Towns	20	20	20
Observations	1827	1827	1827

Notes: Fixed effects regressions with 40 (town by sex) fixed effects. 't' statistics in parentheses are computed from robust standard errors clustered by the 40 groups.

Table 4: Explaining the Heights of 10-13 Year Olds

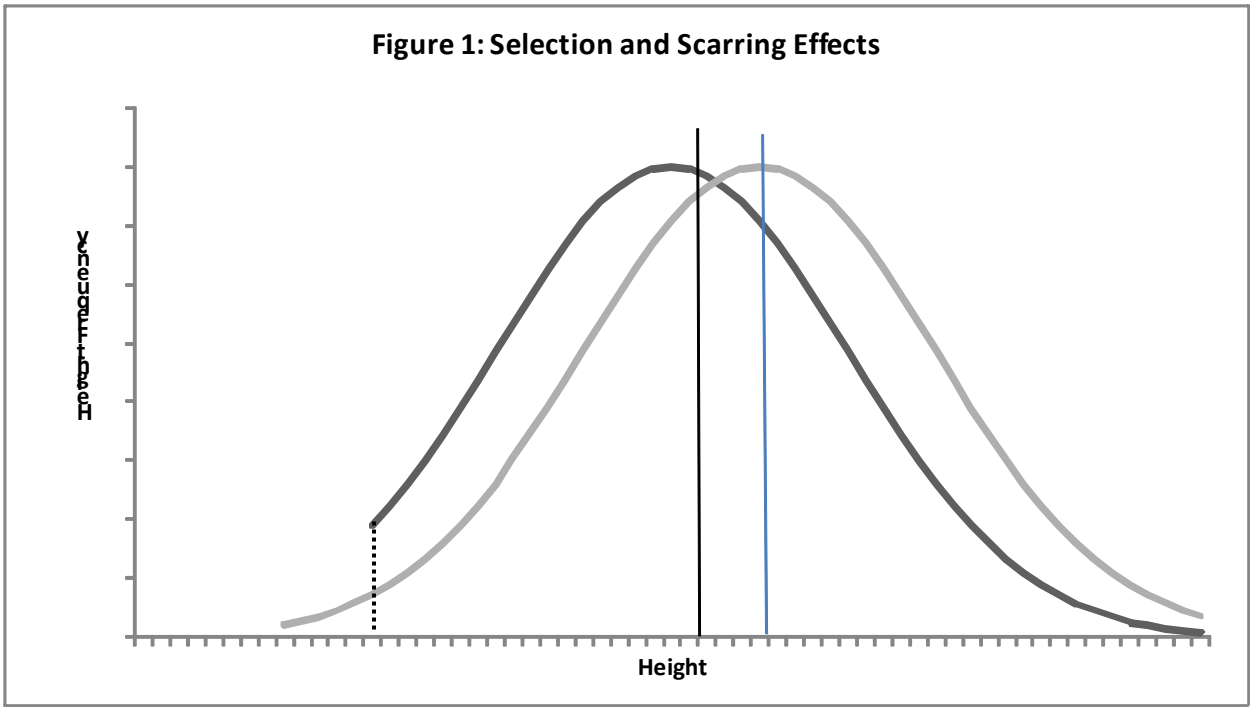
	(1)	(2)	(3)
Infant mortality in birth year	-0.003	0.000	
	(0.30)	(0.01)	
Infant mortality when aged 2-6		-0.025	-0.025
		(2.09)	(2.15)
Year	0.270	0.226	0.226
	(11.18)	(7.82)	(9.37)
Age 11	5.009	5.034	5.035
	(17.06)	(18.00)	(17.96)
Age 12	9.322	9.353	9.353
	(24.14)	(24.62)	(24.63)
Age 13	14.116	14.240	14.240
	(32.67)	(35.30)	(35.75)
Part year	3.879	3.903	3.903
	(3.89)	(3.91)	(3.92)
R^2 (within)	0.726	0.726	0.726
Towns	20	20	20
Observations	1627	1627	1627

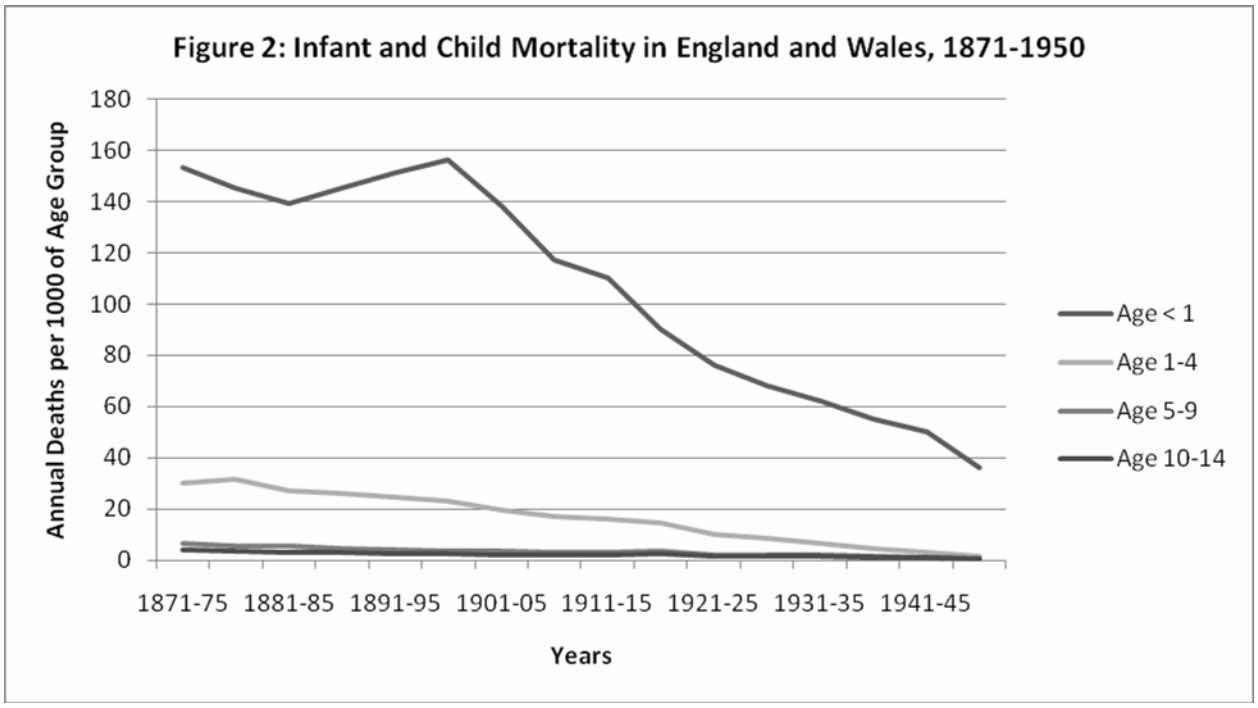
Notes: Fixed effects regressions with 40 (town by sex) fixed effects. 't' statistics in parentheses are computed from robust standard errors clustered by the 40 groups.

Table 5: Regressions by sex and region

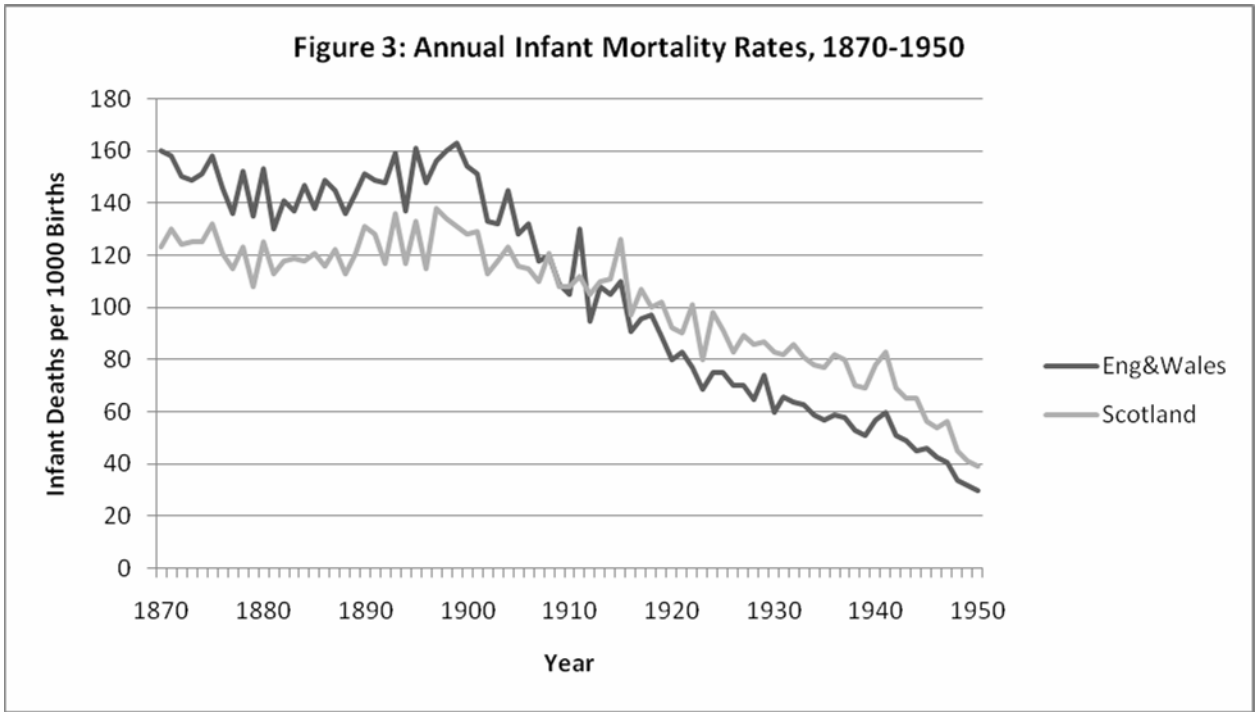
	(1)	(2)	(3)	(4)
	Girls	Boys	North	South
Infant mortality when aged 2-4	-0.035 (3.15)	-0.018 (2.26)	-0.022 (2.93)	-0.035 (2.67)
Year	0.177 (6.62)	0.213 (7.87)	0.207 (10.26)	0.170 (8.97)
Age 7	5.992 (21.90)	5.295 (11.84)	5.618 (19.59)	5.919 (20.00)
Age 8	11.558 (51.10)	11.001 (19.31)	11.271 (31.36)	11.836 (20.47)
Age 9	15.773 (57.03)	15.446 (44.79)	15.538 (68.96)	15.835 (68.51)
Age 10	21.638 (50.90)	20.555 (50.48)	20.981 (58.10)	21.732 (68.51)
Age 11	26.965 (110.49)	25.227 (56.10)	25.897 (72.37)	27.037 (33.98)
Age 12	32.009 (87.73)	29.234 (57.39)	30.347 (70.33)	31.968 (33.98)
Age 13	36.682 (87.73)	33.81 (103.77)	35.031 (88.77)	36.408 (29.88)
Part year	4.591 (6.07)	4.400 (5.98)	4.715 (7.01)	3.404 (5.91)
R^2 (within)	0.958	0.930	0.937	0.975
Towns	20	20	12	8
Observations	1770	1772	2952	590

Notes: Fixed effects regressions with 20 (town) fixed effects in cols (1) and (2) and 40 (town by sex) fixed effects in cols (3) and (4). 't' statistics in parentheses are computed from robust standard errors clustered by the 20 and 40 groups respectively. The total number of observations used in these regressions is slightly larger than in Tables 3 and 4 as they includes some cases for which infant mortality in the year of birth is not available.

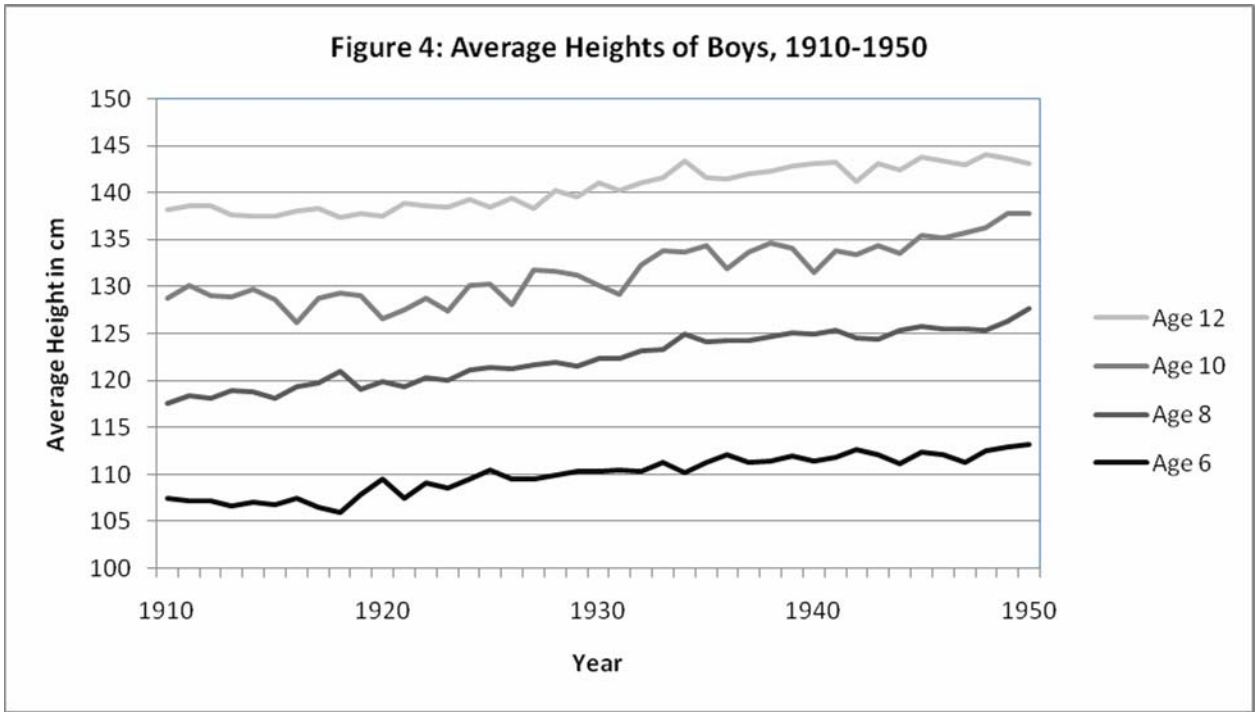




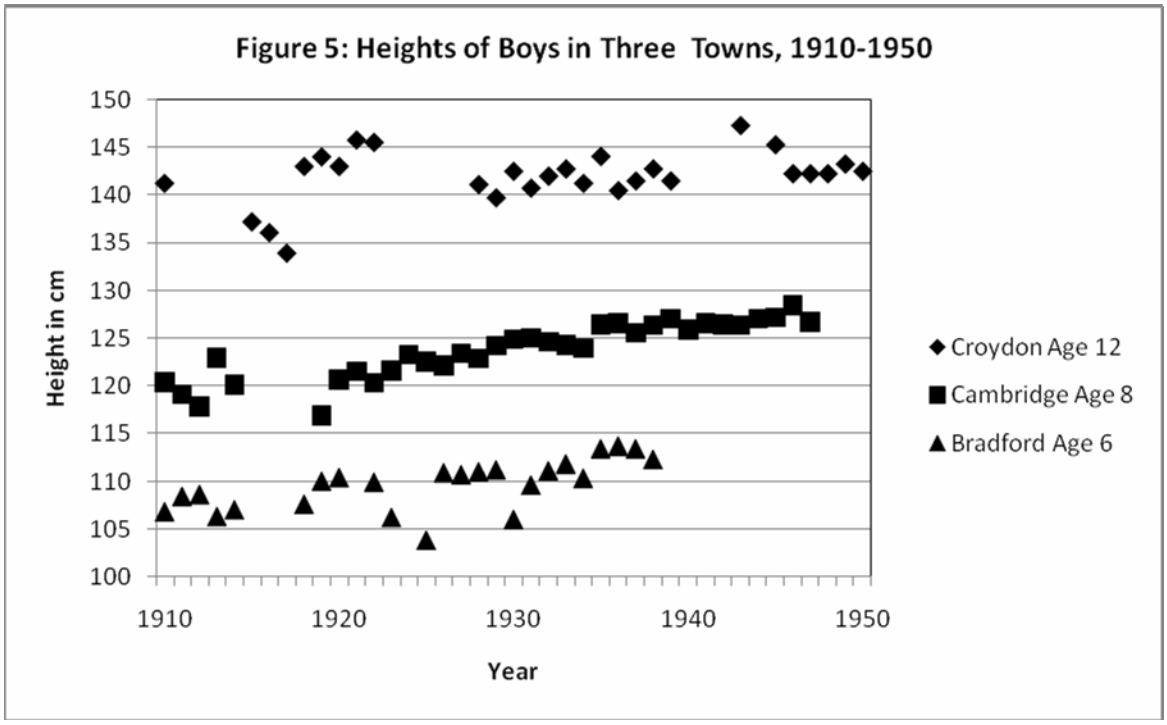
Source: Registrar General, '*Mortality statistics*', Table 4, p. 5.



Source: Mitchell. '*British historical statistics*', pp. 58-9.



Source: See text



Source: See text.